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Stability and Change of Neuroticism in Aging

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Data from the Longitudinal Aging Study Amsterdam were used to study the relationship between neuroticism and aging. At baseline, cross-sectional analyses of data from 2,117 respondents (aged 55–85 years, *M* = 70) showed no significant age differences. The magnitude of the 3- and 6-year stability coefficients was high, and 12% of the elderly participants showed a clinically relevant mean level change. Longitudinal multilevel analyses showed a small but statistical significant change with aging, but the mean change was not considered clinically relevant. A U-formed course was found, showing a slight decrease until respondents reached the age of 70. Adjusting the model for physical health-related variables slightly increased the stability. An additional interaction analysis showed that the individual trajectory of neuroticism was not affected by the physical health status. In conclusion, neuroticism remains rather stable in middle and older adulthood, with some apparent increase in late life.

DOES aging only alter us physically or does our personality change as well? During the past three decades, the question of whether personality changes in later life has been the subject of debate in personality research. Some researchers maintain that personality is fully developed by the time a person reaches the age of 30 and that it becomes increasingly stable with time (Costa & McCrae, 1988, 1994; Costa et al., 2000; Glenn, 1980; Martin, Long, & Poon, 2002; Roberts & Del Vecchio, 2000). Other researchers advocate the plasticity of personality as a function of contextual variables and compensatory behavioral changes to biological aging (Alwin, 1994; Baltes, Staudinger, & Lindenberger, 1999; Caspi & Roberts, 1999; Heatherton & Nichols, 1994; Roberts, 1997). In the present study we aim to examine the cross-sectional and longitudinal stability or change of the personality trait of Neuroticism over a 6-year period of time in a large community sample of Dutch elderly individuals aged between 55 and 85 years at baseline. We hypothesize that we will find general stability, with a minority showing significant changes in neuroticism scores in late life.

In general there are two different approaches to the study of personality stability (Costa & McCrae, 1994): stability of mean levels to estimate aggregate level changes in personality with age, and stability approached as an individual-differences phenomenon, assuming that some people change whereas many remain stable (Cattell, 1950, 1966; Mroczek & Spiro, 2003). A large body of longitudinal research on mean-level changes emphasizes the invariance of neuroticism in late life (Costa & McCrae, 1980, 1990, 1994; Martin et al., 2002; Smith & Baltes, 1999; Watson & Clark, 1984). However, recent results suggest that changes can be observed in old age (Small, Hertzog, Hultsch, & Dixon, 2003).

Studies on individual differences in change are scarce. A recent study by Mroczek and Spiro (2003) found significant variability in neuroticism with age. The younger elderly persons and men who recently experienced a life event showed a sharp decline in neuroticism over the follow-up period.

Neuroticism is a major personality trait (Eysenck, 1990) and one of the most well-established dimensions in the "Big Five" (Costa & McCrae, 1994). Research has shown personality in late life, and neuroticism in particular, to be strongly related to mental and physical health, level of social support, self-rated health, and functional limitations (Duberstein et al., 2003; Hooker, Monahan, Bowman, Frazier, & Shifren, 1998; Siegler & Brummett, 2000; Smith & Gallo, 2001; Watson & Tellegen, 1985). So, age-graded deteriorations in physical health and related declines in daily functioning may affect the association between aging and neuroticism. We hypothesize that these variables affect the individual trajectories of neuroticism in an aging population.

For several reasons, this study is rather unique. To our knowledge, this is the first study to analyze this association in a large community sample of elderly persons between the ages of 55 and 85 years at inclusion. Until now, most research has been performed cross-sectionally or on specific populations. To analyze the developments of neuroticism in aging populations, a prospective design is required. As far as we know, this study is the first using a 6-year longitudinal design with two follow-up measurements in a community population of elderly individuals between 55 and 85 years of age. In addition to this, the multidisciplinary design of this study allowed us to address the influences of physical health-related variables on this association. Finally, we are, to our knowledge, the first researchers looking at individual differences in change on the longitudinal trajectory of neuroticism in a large community sample of elderly people.

Methods

Sampling and Procedures

The present study is part of the Longitudinal Aging Study Amsterdam (LASA). The LASA is an ongoing multidisciplinary study on predictors and consequences of changes in physical, cognitive, emotional, and social functioning in older people in The Netherlands (Deeg, Knipscheer, & van Tilburg, 1993). Full details on sampling and response are described elsewhere (Beekman et al, 1995; Penninx et al., 1997). In short, we drew a random sample of older (55–85 years) men and women from the population registers in eleven municipalities in three geographical regions of The Netherlands. We constructed the sample so as to reflect the national distribution of urbanization and population density. In order to be able to study age and gender differences, we stratified the sample for age and gender. We excluded persons aged 85 and older from the baseline sample, because the attrition rate of this age group was expected to be high over the 10-year study period. We weighted the baseline sample according to expected mortality in each age group, resulting in an overrepresentation of older age strata and a roughly even distribution of men and women. The respondents were visited at home by trained interviewers who worked with laptop computers. Interview and tests took approximately 1.5 hr. In the baseline LASA interview, which took place in 1992–1993, there were 3,107 participants. The first follow-up measurement was performed in 1995–1996 ($n =$ 2,545, 82%), and the second in 1998–1999 ($n = 2{,}076$, 67%).

In the LASA interview, we measured the personality factor Neuroticism by using a self-report questionnaire. The baseline LASA cycle consisted of a face-to-face main interview, after which the interviewer left a questionnaire to be returned by mail. Not all respondents complied, and to be eligible for inclusion in the statistical analyses of the present study, the respondents should have answered more than 80% of all the items on the questionnaire. This cutoff point resulted in a sample of 2,117 participants at baseline (68.3%). The older old ($\chi^2 = 158.13$; $p < .001$) and less educated ($\chi^2 = 41.55$; $p < .001$) were more often lost through item nonresponse. Non-responders had a higher number of chronic diseases ($\chi^2 = 12.7$; $p < .001$), were more often cognitively impaired ($\chi^2 = 217.17$; $p < .001$), and showed more difficulties while performing activities of daily living ($\chi^2 = 74.26$; $p < .001$).

For the multilevel analyses, we used 2,117 cases at baseline, and 1,983 and 1,647 cases at first and second follow-up measurement. Data were available for 1,229 respondents on all three measurements. These 1,229 respondents had a higher mean age ($\chi^2 = 328.86$; $p < .001$), and they were more often male ($\chi^2 = 4.827$, $p < .05$) and higher educated ($\chi^2 = 13.62$, $p < .001$). They reported fewer chronic diseases ($\chi^2 = 49.23$, $p < .001$) and functional limitations ($\chi^2 = 118.306$, $p < .001$) at baseline in comparison with those respondents lost during follow-up. Finally, respondents lost during follow-up were more often cognitively impaired ($\chi^2 = 83.25$, $p < .001$), but we found no significant difference in mean neuroticism level at baseline ($\chi^2 = 1.49$, $p = .22$).

Measurements

Neuroticism.—We operationalized neuroticism by using a subset of 25 items out of a list of 36 neuroticism items from the Dutch Personality Questionnaire (DPQ; Luteijn, Starren, & van Dijk, 1975, 2000). The pilot studies of the LASA program showed that the original 36-item scale measuring neuroticism can be abbreviated, because the original DPQ scale contained items that were less valid for an aging population and contained too many items for administration in older populations (de Beurs, Beekman, Deeg, van Dyck, & van Tilburg, 2000; Smits, Deeg, & Bosscher, 1995; Steunenberg, Beekman, Deeg, & Kerkhof, 2003). Persons scoring high on neuroticism items experience a broad range of negative moods, including emotions such as fear, sadness or depression, and self-dissatisfaction. High scorers are socially ill at ease and feel they cannot easily relate to other people, experience an inability to inhibit cravings, and are more vulnerable to stress (Costa & McCrae, 1980, 1984a; Luteijn et al., 2000). An example of a neuroticism item is "I often hate myself." Interviewers asked respondents to indicate whether various similar statements applied to them (yes = 2, do not know = 1, no = 0). Total scores range between 0 and 50. These DPQ items have strong negative relations with the Emotional Stability scale of the Revised NEO Personality Inventory (NEO-PI-R; Luteijn et al., 2000). We tested the utility and psychometric properties of the shortened scale, and it appeared to be a reliable (Cronbach's $\alpha = .86$) and valid instrument to measure neuroticism in the elderly population (Steunenberg et al., 2003).

Independent variables.—We analyzed demographic variables and physical health-related variables for their association with stability or change of the level of neuroticism. We determined the presence of chronic diseases by asking the respondents whether they had any of the following diseases: cardiac disease; peripheral atherosclerosis of the abdominal aorta or the arteries of the lower limb; stroke; diabetes mellitus; lung disease (asthma or chronic obstructive pulmonary disease); malignant neoplasms; arthritis (rheumatoid arthritis or osteoarthritis); or any other major chronic diseases (Central Bureau of Statistics, 1989). We calculated the number of chronic diseases by summing up all specific diseases reported to be present. In a validation study, respondents' self-reports were compared with information obtained from their general practitioners, and this proved to be sufficiently reliable (Kriegsman, Penninx, Eijk, Boeke, & Deeg, 1996). For the present study, the presence of chronic diseases was indicated as 0 = no disease, 1 = one disease, and 2 = more than one disease.

We measured cognitive functioning by means of the Mini-Mental State Examination (MMSE; Folstein et al., 1975), a frequently used screening instrument for global cognitive dysfunctioning. On 23 questions and tasks, respondents received 1 or more points when they gave the correct answer or performed the task correctly. Scores ran from 0 (all answers incorrect) to 30 (unimpaired). We divided respondents into two categories: MMSE < 24 (impaired cognition, 0) and MMSE $\geq$ 24 (normal cognition, 1).

We assessed functional limitations by asking the respondent the degree of difficulty he or she had with the following activities of daily living: climbing up and down a staircase of 15 steps without stopping, cutting one's own toenails, and using public transportation (Kriegsman, van Eijk, & Penninx, 1997; van Sonsbeek, 1988). Response categories were "yes, without difficulty," "yes, with difficulty," "only with help," and "no, I cannot." The number of limitations ranges from zero (low) to three (high). For this study, the presence of functional limitations was indicated as 0 = no functional limitations, 1 = one functional limitation, and 2 = more than one functional limitation.

The demographic variables included age group, gender, and level of education. Age ranged between 55 and 85 at baseline;

for the purpose of data analyses, we categorized age in three age groups: 55–64, 65–74, and 75–85. We measured education on a 3-point ordinal scale: low (elementary or lower vocational education = 1), middle (general education = 2), and high (higher vocational, college, or university = 3).

Data Analysis

We analyzed data in three steps. First, we tried to find significant predictors of mean neuroticism level at baseline. To assess cross-sectional differences in the mean levels of the neuroticism scores, we performed univariate analyses of variance (ANOVA). In order to attain the most parsimonious set of predictors, we performed a forward-stepwise regression analysis. Although the observations on the neuroticism items were not normally distributed, because of the large number of observations the skewness had only a slight effect on the power of the ANOVAs and regression analysis (central limit theorism; see Bock, 1975).

Second, we assessed longitudinal changes in mean neuroticism score. To find out how many respondents show a clinically relevant change in mean neuroticism level during follow-up, we used a relevant change criterion. Clinical significance refers to the practical value or importance of the effect of an intervention, that is, whether it makes any real difference to the clients or to others in their functioning and everyday life. We defined a relevant clinical change as a decrease or increase of at least 8 points on the DPQ between two measurements. The difference of 8 points represents, in clinical terms, a large change (Lipsey & Wilson, 1993). It is greater than 5, which, on this scale, corresponds to the threshold for statistically significant change (Drenth, 1972; Jacobsen & Truax, 1991).

The third data analytic approach that we used in this study was multilevel analysis. We investigated the longitudinal stability or change of neuroticism with aging by using a multilevel analysis (also known as random coefficient analysis; see Goldstein, 1995; MlwiN, version 1.10.0007; Centre for Multilevel Modeling, Institute of Education, London). We performed this type of analysis because this statistical technique makes it possible to analyze longitudinal relationships by using all available longitudinal data, without summarizing the longitudinal development of each subject into one value. In contrast to the more traditional methods of longitudinal data analysis (i.e., a multivariate analysis of variance, or MANOVA, for repeated measures), which require a complete longitudinal data set, in a multilevel analysis, both the number of observations per individual as well as the time interval between observations may vary. Multilevel analysis does not use strict conditions concerning the type of missing data, because it assumes data missing at random: that is, given the observed data, the unobserved data are random.

In the present study, we defined a two-level hierarchy to form random regression models to describe the individual variability in the longitudinal development of neuroticism in an aging population. The first level is defined by age (range 55–85) and the second level by the respondents. Because the neuroticism score was highly skewed, we performed the longitudinal analyses by using a logarithmic transformation.

We entered both the outcome variable of neuroticism as well as age as continuous variables. We included age in all models, in order to test the influence of aging on the neuroticism score. We tried to find the best model for neuroticism in aging. After assessing the "crude" linear association of neuroticism with aging, we added a quadratic age effect to the model (Model 1).

Model 1:

$$\ln(\text{neuroticism score}) = \beta_{0ij} + \beta_{1ij}\ \text{age} + \beta_{2ij}\ \text{age}^2,$$
$$\beta_{0ij} = \beta_0 + u_{0ij} + e_{0ij}.$$

We adjusted the statistical model by adding covariates, considering first gender, level of education (because level of education is a variable with three categories, two dummy variables were entered to the equation), number of chronic diseases, functional limitations, and cognitive functioning in order to analyze whether these variables influence the longitudinal development of neuroticism.

Model 2 (adjusted for all covariates):

$$\begin{aligned}\ln(\text{neuroticism score}) &= \beta_{0ij} + \beta_{01ij}\ \text{age} + \beta_{02ij}\ \text{age}^2 + \beta_{03}\ (\text{gender}) \\ &\quad + \beta_{04}(\text{middle level of education}) \\ &\quad + \beta_{05}(\text{high level of education}) \\ &\quad + \beta_{06}(\text{chronic diseases}) + \beta_{07}(\text{functional limitations}) \\ &\quad + \beta_{08}(\text{cognitive functioning}),\end{aligned}$$
$$\beta_{0ij} = \beta_0 + u_{0ij} + e_{0ij}.$$

In the third step, to identify significant predictors of individual differences in change, we entered the interaction terms between the physical health-related variables and age into the model.

Results

Table 1 summarizes the demographic and physical health-related characteristics of the baseline sample. At inclusion, the mean age of the 2,117 respondents was 70 years; 51% were female. The age group and gender distributions are the results of the stratified sampling. The high number of participants with a chronic illness (60%) or physical functional limitations (41%) is a function of the oversampling among the older old. It demonstrates that attrition has not led the participants to become a sample of "healthy elderly" persons. Table 1 contains the distribution of average neuroticism scores by demographic and physical health-related variables, and the results of the univariate ANOVA are summarized in this table.

We found no significant association between age group and the mean score on neuroticism ($p = .158$). The age groups do show an increase in mean level of neuroticism with age, but this linear association was not significant. Women scored significantly higher on the neuroticism factor ($p < .001$), and respondents with a middle or higher education scored significant lower than the lower educated respondents ($p < .001$). The cognitively impaired respondents (MMSE ≤ 24) showed a significant higher level of neuroticism than those with a normal cognitive functioning ($p < .01$). We found significant associations with the number of chronic diseases ($p < .001$) and functional limitations ($p < .001$). The more chronic diseases and the more functional impaired, the higher the score on the neuroticism questionnaire was.

The results of the multiple linear regression analysis are shown in Table 2. The regression equation with four of

Table 1. Neuroticism Scores at Baseline Against Demographic Characteristics and Health-Related Variables

Characteristic	n	%	Avg. Neuroticism Score M	(SD)	F
Score neuroticism	2,117	100	11.70	(8.85)	
Age group (years; M = 69.5)					
55–64	774	37	11.30	(8.50)	$F_{2,\ 2114} = 1.85$
65–74	711	33	11.73	(9.17)	
75–85	632	30	12.21	(8.93)	
Gender					
Male	1,042	49	10.66	(8.85)	$F_{1,\ 2115} = 29.8$***
Female	1,076	51	12.75	(8.76)	
Education					
Low	1,259	59	12.64	(9.34)	$F_{2,\ 2112} = 18.5$***
Middle	592	28	10.62	(7.93)	
High	265	13	9.70	(7.75)	
No. of chronic diseases (M = 0.93)					
None	852	40	10.47	(8.38)	$F_{2,\ 2113} = 27.9$***
One	759	36	11.81	(8.33)	
More than one	505	24	13.69	(10.00)	
General cognitive functioning (M = 27.3)					
MMSE < 24	145	7	13.41	(9.36)	$F_{1,\ 2109} = 5.76$*
MMSE 24	1,966	93	11.59	(8.80)	
Functional limitations (M = 0.64)					
None	1,793	59	10.38	(8.34)	$F_{2,\ 2100} = 44.3$***
One	584	19	13.22	(8.92)	
More than one	685	22	14.74	(9.51)	

Note: MMSE = Mini-Mental State Examination (score).
*$p < .05$; **$p < .01$; ***$p < .001$.

the predictors was significant, $R = .25$, adjusted $R^2 = .06$, $F(4,\ 2088) = 34.32$, $p < .001$. The predictors cognitive functioning ($p = .727$) and age group ($p = .183$) showed no significant relation with neuroticism after the other factors were controlled for. Functional limitations showed the strongest association with the mean level of neuroticism. This predictor alone accounted for 4% of the variance of the baseline level of neuroticism, whereas the other variables contributed an additional 2%.

Preceding the longitudinal multilevel analyses on effects of aging on neuroticism, as a kind of explorative test–retest, we calculated Pearson correlation coefficients between the total mean scores on neuroticism for the three measurements. We deleted respondents listwise, leaving 1,229 respondents who conducted all three measurements. For the period between

Table 2. Predictors of Neuroticism: Multivariate Regression Analysis

Independent Variables	SRC	R Cum	Adjusted R^2 Cum
1. Functional limitations	.145	.199	.039*
2. Level of education	−.093	.226	.050*
3. Gender	.081	.239	.056*
4. No. of chronic diseases	.073	.248	.060*
$F(4,\ 2088) = 34.31$, $p < .001$			

Notes: This is a multiple linear regression; dependent variable of neuroticism (Dutch Personality Questionnaire), n = 2,093. R Cum = cumulative amount of explained variance; SRC = standardized regression coefficient.
*$p < .05$.

Table 3. Changes in Mean Level of Neuroticism Categorized by Effect Size

Effect Size: Δ (Mean Neuroticism Score)	No. of Respondents: Baseline to First Follow-Up	Baseline to Second Follow-Up
Great decline (δ ≥ 0.9)	57 (4.6)	57 (4.6)
Medium to great decline (0.9δ ≤ 0.5)	83 (6.8)	99 (8.1)
Small to medium decline (0.5δ ≤ 0.2)	136 (11.1)	159 (12.9)
No change	552 (44.9)	543 (44.2)
Small to medium increase (0.2δ ≤ 0.5)	198 (16.1)	174 (14.2)
Medium to great increase (0.5δ ≤ 0.9)	107 (8.7)	116 (9.4)
Great increase (δ ≥ 0.9)	96 (7.8)	81 (6.6)

Notes: n = 1,229. Baseline to first follow-up, mean interval = 3 years; baseline to second follow-up, mean interval = 6 years. Great decline and great increase show a clinically significant change. Percentages are shown in parentheses.

baseline and first follow-up (mean interval of 3 years), we found a coefficient of .75; for the period between baseline and the second follow-up (mean interval of 6 years), we found a coefficient of .77 (results not shown). These test–retest coefficients are rather high and stable over the 6-year follow-up period, indicating a stability of test scores over time. We adjusted the coefficients for baseline age, gender, level of education, number of chronic diseases, functional limitations, and cognitive functioning. The associations were hardly affected by these adjustments and remained stable. The coefficients ranged between .73 and .77.

In Table 3, we present the neuroticism mean score differences between baseline and first or second follow-up, answering the question of how many respondents show a clinically significant change. We categorized respondents by the strength of the effect size of this mean-level change. A large majority of nearly 90% shows a small to medium effect size (δ ≤ .90), indicating stability. A great effect size (difference of more than 8 points) is seen as a significant clinical change in mean neuroticism level; 4.6% of the respondents between baseline and first follow-up or second follow-up show a clinically significant decrease, and 7.8% of the respondents between baseline and first follow-up and 6.6% of the respondents between baseline and second follow-up show a clinically relevant increase of neuroticism level.

Table 4 presents the coefficients and standard errors from multilevel models of individual differences in the longitudinal development of neuroticism in an aging population. We found all coefficients to be significant ($p < .05$). Table 4 should be interpreted as follows: for any specific age one can, by means of the coefficients and random variances, compute the sum of the regression equations, and this sum has to be returned to the base-e logarithmic (ln) to get the mean neuroticism score for this specific age. The results as presented in Table 4 are depicted in Figure 1. This figure shows the crude and adjusted longitudinal development of the mean neuroticism score in the research population aged between 55 and 85 years. The longitudinal multilevel analyses report a statistically significant change in mean neuroticism level with age. The figure depicts a U-formed shape, showing a slight decrease until the age of 70 (1.5 DPQ points), after which a slight increase is found until the age of 85 (increase of 1.5 DPQ points). The mean scores at age 55 and 85 are nearly the same. Adjusting for age group, gender,

Table 4. Coefficients and Standard Errors From Multilevel Models of Individual Differences in the Longitudinal Development of (ln) Neuroticism in an Aging Population

Variable	Unadjusted (Model 1)	Model Adjusted for Gender (Model 2)	Level of Education	No. of Chronic Diseases	No. of Functional Limitations	Cognitive Functioning	Model Adjusted for all Covariates
Fixed parameters							
Intercept, β_{0ij}	5.86 (.82)	5.30 (.81)	5.64 (.79)	5.59 (.78)	5.52 (.82)	5.75 (.81)	5.31 (.80)
Age, β_{01}	−0.11 (.02)	−0.10 (.02)	−0.10 (.02)	−0.10 (.02)	−0.09 (.02)	−0.10 (.02)	−0.09 (.02)
Age^2, β_{02}	0.0008 (0.0002)	0.0007 (0.0002)	0.0007 (0.0002)	0.0007 (0.0002)	0.0007 (0.0002)	0.0007 (0.0002)	0.0006 (0.0002)
Gender, β_{03}		0.24 (0.03)					0.20 (0.35)
Middle level of education, β_{04}			−0.12 (.04)				−0.10 (0.04)
High level, β_{05}			−0.17 (0.05)				−0.11 (0.05)
No. of chronic diseases, β_{06}				0.05 (0.01)			0.03 (0.01)
No. of functional limitations, β_{07}					0.09 (0.02)		0.06 (0.02)
Cognitive functioning, β_{08}						−0.04 (.05)	−0.01 (0.04)
Random variances							
Intercept, u_{0ij}	0.65 (0.02)	0.63 (0.02)	0.64 (0.02)	0.64 (0.02)	0.63 (0.02)	0.65 (0.02)	0.61 (0.02)
Error, e_{0ij}	0.25 (0.01)	0.25 (0.01)	0.25 (0.01)	0.25 (0.01)	0.25 (0.01)	0.25 (0.01)	0.25 (0.01)

Note: no significant changes of the models were observed by adding random variances for age and age^2.

level of education, cognitive functioning, and number of chronic diseases and functional limitations hardly influenced the development of neuroticism in aging, and in general little to no change in mean neuroticism level is shown in late life. The mean-level change for the total sample is about 2 points, so using the relevant change criterion, we find that the mean-level change is not clinically significant. Because we tried to model the longitudinal development of neuroticism in an aging population, the range of mean neuroticism scores, as presented in Figure 1, decreases if compared with the cross-sectional baseline range as presented in Table 1.

In the final analyses step, we added the interaction terms of physical health-related variables to find out if these variables were significant predictors of individual differences in change. The variables entered were cognitive functioning, number of chronic diseases, and number of functional limitations. All three variables were not associated with individual differences in changes of neuroticism level. Therefore, the individual longitudinal trajectory of neuroticism in late life is not affected by the number of chronic diseases, functional limitations, or level of cognitive functioning.

Discussion

The overall conclusion that can be drawn from this study is that, for a majority, neuroticism remains rather stable in old age; for those who do change, the level of change is not affected by the deteriorations of physical health or cognitive functioning known to be related to aging. Although we hypothesized that we would find an increase of the mean level with aging, on baseline we found no significant association between age group and the mean score of neuroticism. In addition, in the multiple regression analyses, we found age group not to be associated with neuroticism level. Thus, at a cross-sectional level, we did not find a significant association between aging and neuroticism, indicating a stability of mean neuroticism level. In addition to this, the absolute magnitude of the test–retest coefficients in this study was rather high (.75), even when we adjusted it for demographic and physical health-related variables found to be related to the mean level of neuroticism, indicating a stability in neuroticism score over time. These coefficients were similar to those reported elsewhere, and which are seen as evidence for the stability assumption (Costa & McCrae, 1988).

However, the longitudinal multilevel analysis reports a statistically significant change with aging. We found a U-formed course, showing a slight decrease until the age of 70 and a slight increase after this age. This U-formed course is very hard to explain. In general there are two possible explanations for changes in mean score in late life. The first possible explanation is a survivor effect. The longitudinal results indicate an effect of physical health on the stability of neuroticism, whereas adjusting the regression model for number of functional limitations slightly increases the stability of the development of neuroticism in an aging population. The more frail elderly individuals were more often lost by attrition. The respondents who were lost by attrition during follow-up showed significantly more difficulties while performing daily living activities. Costa & McCrae (1985) show that respondents who have a higher number of chronic diseases or functional limitations are higher in neuroticism. The decline in mean level of neuroticism might be explained by the attrition of the most frail elderly individuals, suggesting that those respondents who continue the study are the more healthy respondents. The slight increase after the age of 70 may be explained by an aging effect. Aging is known to be associated with an increase of physical illness and associated functional limitations, and a decrease of social resources. These factors are also known to be related with the mean neuroticism level (Costa and McCrae, 1985, 1988).

The mean neuroticism-level change for the total sample found in the longitudinal multilevel analyses was about 2 DPQ points, which proved to be statistically significant, but it is rather small and, according to our relevant change criterion, cannot be seen as a clinically significant change (Drenth, 1972; Jacobsen & Truax, 1991; Lipsey & Wilson, 1993).

The degree of change and the impact of that change are important for deciding clinical relevance. Merely examining the absolute level of change is not always enough to determine whether the difference or change really makes a difference.

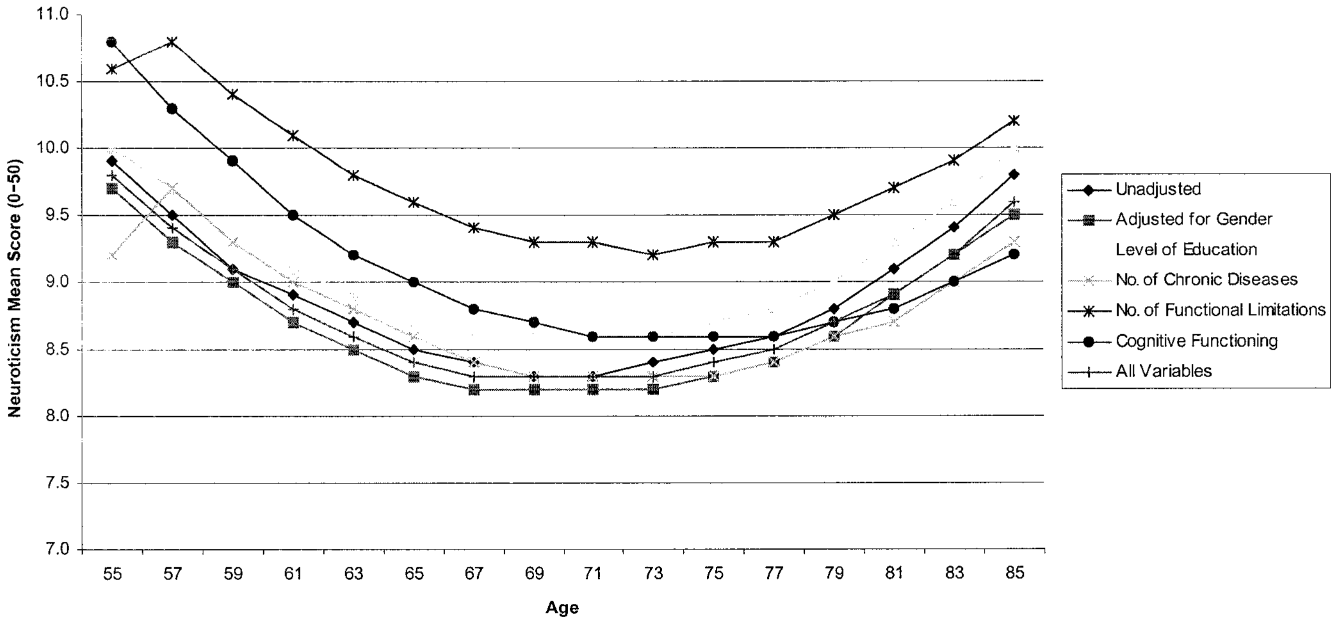

Figure 1. Longitudinal course of mean neuroticism scores in aging, adjusted for confounders.

A mean change of 2 points on a scale with a maximum score of 50 is small, and the impact on everyday performance is negligible. Preceding these multilevel analyses, we analyzed how many respondents did change, using a clinically relevant change criterion. Of all the respondents, 5% showed a clinically significant decrease and 7% an increase.

Finally, we searched for significant predictors of individual differences in changes in neuroticism trajectories. None of the physical health-related variables were associated with individual differences in neuroticism trajectories. In other words, for those respondents who do change, the mean level of neuroticism is not affected by the number of chronic diseases, functional limitations, or level of cognitive functioning. A recent study by Mroczek and Spiro (2003) also found no significant association for functional limitations.

The strengths of this study are the large study population ($n = 2{,}117$) and the prospective follow-up design. The present study is one of the first 6-year longitudinal designs, investigating age changes and differences in the personality dimension neuroticism in a large, representative population of elderly persons. Because of the multidisciplinary design of the LASA study, we found it not only possible to examine the relationship between the level of neuroticism and the demographic characteristics, but we could also study whether physical health-related characteristics did influence the stability of neuroticism in the aging.

A limitation of the present study is the large number of respondents lost by attrition (31.7%). As in all community-based studies of elderly persons, the older old and less educated, those with cognitive impairments, and the respondents with more chronic diseases and difficulties while performing daily living activities were more often lost through attrition, which may threaten the generalizability of the results.

In summary, we can conclude that neuroticism remains rather stable in old age. In the cross-sectional analyses, age shows no significant association with neuroticism. The magnitude of the stability coefficient is high and in line with findings supporting the stability assumption (Costa & McCrae, 1988, 1994); 12% of the elderly individuals show a clinically relevant change. Finally, the longitudinal change found to be statistically significant cannot be considered clinically relevant (Drenth, 1972; Jacobsen & Truax, 1991; Lipsey & Wilson, 1993). In addition to this, we hypothesized that we would find significant influences of age-graded deteriorations in physical health and related declines in daily functioning on individual trajectories of neuroticism. However, our results showed that individual differences in change of neuroticism in late life are not affected by individual differences in physical health-related variables. In conclusion, neuroticism remains rather stable in very old age; although we did find significant individual differences in trajectories, we could not explain these differences by late-life increase in physical illness and associated functional limitations.

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